

Contribution of Lead in Dust to Children's Blood Lead

by Michael J. Duggan*

The importance of urban dust as a source of lead for young children is still disputed. Although blood-lead data from various population surveys usually show a peak concentration in early childhood, there is evidence that such a peak is small or absent altogether in children without much access to the general environment. An examination of those studies where groups of people in regions of low and high lead contamination have been compared shows that the child/adult blood-lead ratio is almost always enhanced in the more exposed groups. This implies a route of lead uptake which is important for children but less so for adults, and it is likely that this route is the dust-hand-mouth one. There are sufficient data to suggest a quantitative relationship between raised levels of blood lead and lead in dust. There is a strong case for a lead-in-dust standard but some will probably remain unpersuaded unless or until there are reliable data for blood lead and environmental lead involving matched groups of young people from urban and rural areas.

Introduction

Several authorities have set standards or guidelines for community exposure to airborne lead. These imply, and in some cases have been explicitly derived from, maximum allowable increases in blood-lead levels. For example, the Commission of the European Communities proposed standard of $2 \mu\text{g}/\text{m}^3$ has been based on the premise that the contribution made by inhaled lead to the blood-lead level should not exceed $5 \mu\text{g}/\text{dL}$ (1). However, inhalation not the only route by which people take in lead from the environment. Many authors have suggested that the unwitting ingestion of dust via dirty hands gives rise to a significant lead intake, particularly for young children. If this is so, then it would seem reasonable to define a standard in terms of any combination of airborne lead and lead in dust which brings about some maximum allowable increase in blood lead level (2, 3).

Not all agree with the view that lead in dust represents an important source of lead for urban children. Studies showing the variation of blood-lead level with age usually show a peak value at 2 to 3 years, and Ter Haar (4) attributes this to the gnawing of paintwork by children in this age group.

Chamberlain et al. (5) have pointed out that several surveys of blood-lead levels in children and their mothers show child/mother ratios in the region of 1.3. Further, blood-lead levels are almost always found to be higher in adult males than females; the male/female blood-level ratio is often rather more than 1.3. So the blood-lead levels of young children appear to be intermediate between those of their parents. Chamberlain comes to the conclusion (personal communication) that for urban areas—excluding the immediate vicinity of smelters—there is no evidence of an important pathway of lead uptake which affects children but not adults. The UK Department of Health and Social Security Working Party on environmental lead (6) apparently reached the same conclusion, since they ignored any uptake from dust and dirt in their estimates of the contributions of various sources of lead to the body burden of inner-city children. However, there are data from several types of study which suggest that lead in dust makes a significant contribution to the blood lead of urban children.

Effects of Living in a Contaminated Area

A number of studies have been reported in which the blood-lead level of children appears to be related to the level of lead contamination in their environment. The source of contamination is usually a

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smelter, or an occupationally exposed parent, a central urban area or a naturally occurring high concentration of lead in soil. The usual approach of the investigators is to identify two (sometimes more) groups of children who are exposed to different levels of contamination. Measurements are then made of the children's blood-lead levels and of the environmental contamination; besides lead in dust and soil, the environmental measurements usually include airborne lead, and sometimes lead in water and in food.

There are several recent reviews (2, 7) of studies of this kind, and so they will not be discussed in any detail here. The major finding in almost all of them is that the children living in the more contaminated areas have the higher blood-lead levels and that the inhalation of airborne lead is not sufficient explanation. There must therefore be other routes besides inhalation which are important in the transfer of lead from the environment to children and the ingestion of lead in dust via the hands is one obvious possibility.

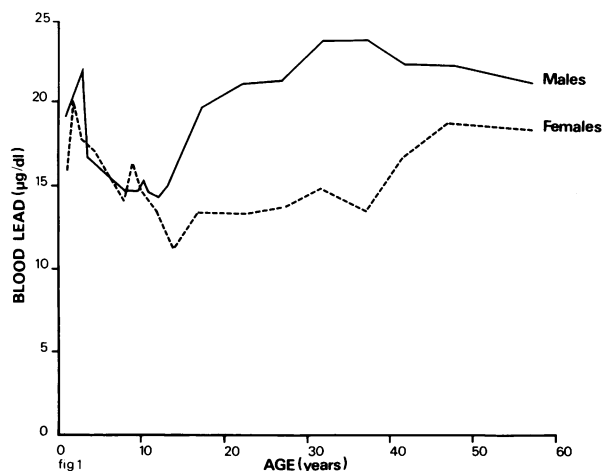


FIGURE 1. Blood-lead levels in Birmingham, UK; from Department of the Environment (7).

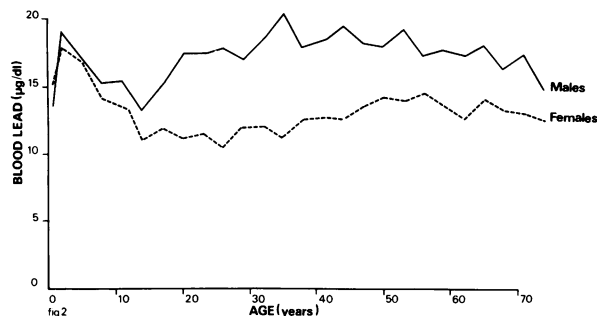


FIGURE 2. Blood-lead levels in the United States; from Mahaffey et al. (8).

The quantitative relationship between children's blood-lead levels and the concentration of lead in dust which can be inferred from these studies is discussed later in this paper.

Variation of Blood-Lead Level with Age

A few surveys of blood-lead levels in a large sample of the general public have been carried out from which some picture of the change in level with age can be obtained. Two such surveys are the one made in Birmingham UK (8) and the one made in the United States as part of the Second National Health and Nutrition Examination Survey (9). The results from these two surveys are summarized in Figures 1 and 2. In spite of major differences between the study populations, there are some similarities in the two sets of results. Both show a peak childhood value occurring at 2-3 years, followed by a decline until early adolescence; there is little or no variation with sex during this period. During adolescence the blood-lead levels of the males, but not the females, rise quite sharply. After adolescence there is a slow rise until middle age, and then some suggestion of gently declining levels throughout the rest of life. A male/female ratio greater than unity persists from adolescence onwards. The blood-lead results from the most recent UK survey [10] are shown in Figure 3 (inner and outer city measurements combined), and give a rather similar pattern; the report on this study does not give the basic data for children but it does contain a graph of blood-lead level (controlled for a number of variables) against age which shows a maximum value at 2 years. Figures 1-3 illustrate the need to define age when referring to the blood-lead levels of a population. For adults in the age group 20 to 40 years and 2-year-old children, some blood-lead ratios of interests are

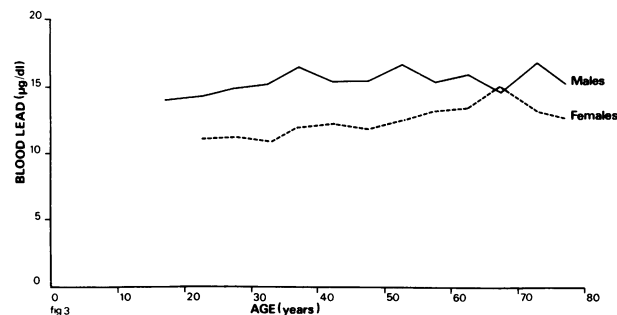


FIGURE 3. Blood-lead levels in the United Kingdom; from Department of the Environment (9).

given in Table 1. It is not clear how the blood-lead level changes during the first year or so of life. The concentration of lead in cord blood is usually slightly lower than that in the mother's blood, and this implies that the initial rise shown in Figures 1 and 2 is the continuation of a rise that started at birth. However, it has been suggested that the blood-lead level actually falls during the first few months of life before beginning to rise. Haas and his co-workers (11) measured the blood-lead levels of several groups of people in a German city: 294 mothers, their newly born infants (by cord-blood samples), 162 hospitalized children aged 8 days to 5 years (those with conditions likely to influence blood-lead levels were excluded), and 201 healthy children aged 6 years to 8 years. The mean blood-lead levels of mothers and their newly born were unremarkable ($16.9 \mu\text{g/dL}$ and $15.7 \mu\text{g/dL}$ respectively) but the levels in the other children were unusual in their very low values during the first year of life, and their steady rise in subsequent years with no evi-

Table 1. Blood-lead ratios from three surveys.

Survey	No. of people examined	Blood-lead ratio	
		Man/woman	Child/woman
Birmingham (1978)	1607	1.56	1.38
NHANES (1979)	4635	1.54	1.55
UK Dept. of Env. (1981)	4981	1.35	—

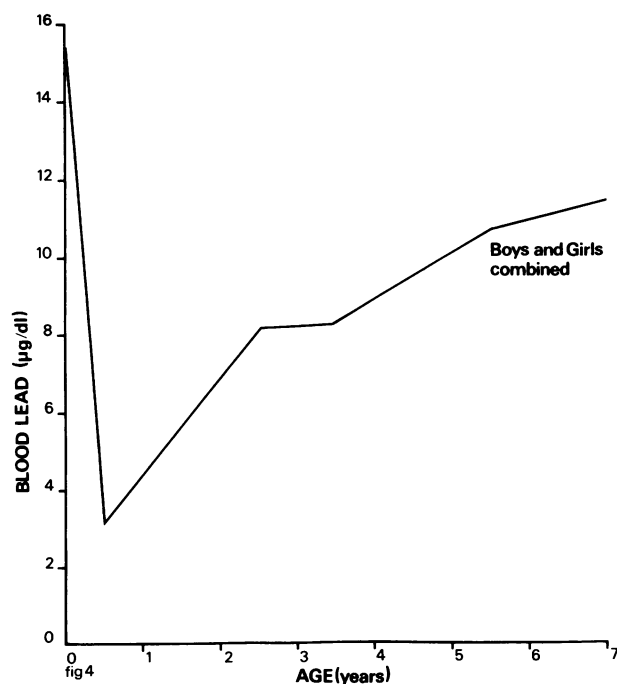


FIGURE 4. Blood-lead levels in newborn, hospitalized, and school children in Nurnberg; from Haas et al. (10).

dence of a peak value during the second or third years. The results are summarized in Figure 4. The ratio of blood-lead levels in 2-year-old children compared with women was about 0.4.

There are other studies which suggest that children isolated from a normal environment show little or no evidence of peak blood-lead levels at 2-3 years. Sartor and Rondia (12) measured lead concentrations in blood collected from blood donors (adults of course) in Liege, and from 127 children (0 to 14 years) in a hospital in the same city. Their results are summarized in Figure 5. It can be seen that the ratio of blood-lead levels in 2 year-old children compared with women is about 0.9. Nygaard et al. (13) looked at the blood-lead levels of 126 young children from seven different areas in Denmark. Their results are summarized in Table 2.

There are some significant differences between the mean blood-lead concentrations of the groups but there is no obvious relationship between age and blood lead. The two groups (K and F) having the lowest blood-lead values might have been expected to be among the highest if there were a peak at 2-3 years. Both these groups were isolated from the general environment insofar as group K were children in hospital, and group F were from a day

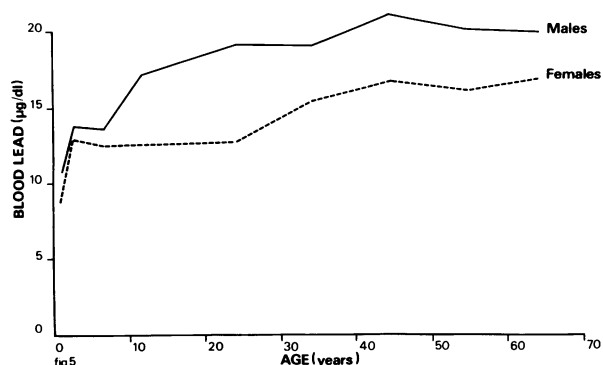


FIGURE 5. Blood-lead levels in adults and hospitalized children in Liege; from Sartor and Rondia (11).

Table 2. Blood-lead levels of Danish children.^a

Group	No. of children	Mean age, yr	Blood-lead level, $\mu\text{g/dL}$	
			Mean	95% confidence interval
A	20	5.0	6.7	± 1.22
G	9	4.8	9.2	± 1.82
D	6	4.6	10.1	± 2.22
B	9	3.9	6.9	± 1.82
K	64	3.8	5.6	± 0.68
C	10	3.7	8.3	± 1.64
F	8	1.8	4.3	± 0.50

^a Data of Nygaard et al. (13).

nursery. The authors (13) comment that "Children of that age at day nursery are protected from contact with the environment compared to the kindergarten children in group G from the same place."

It is worth noting here that blood-lead surveys carried out in Scandinavian countries tend to give a different picture from the UK and US surveys mentioned earlier. Thus, the survey made by Nygaard et al (which included adults as well as children) showed generally low blood-lead levels and a steady increase in the level from birth to old age with no significant differences between males and females; the number of people (about 100) from whom samples were taken was rather small. Relatively low blood-lead levels in adults have also been reported by Nordman (14) and in young children by Bach (15) and by Taskinen et al. (16).

Gibson and her colleagues (17) looked at three groups, each containing 20 children, from Glasgow. All the children were between 3 and 11 years old and matched for age within the groups. Group A was composed of children of normal intelligence, group B of children mentally retarded from known causes (mongolism, phenylketonuria, or severe organic brain damage) and group C of children who were mentally retarded in the absence of any recognized cause. The results of this study are summarized in Table 3. The authors suggest that the relatively low blood-lead levels found in group B may have been due to these children being under closer supervision and being more restricted in their movements than the other two groups.

Zielhuis et al. (18) measured lead concentrations in blood from 48 patients (2 months to 6 years of age) in an Amsterdam hospital. They found a significant positive relationship between age and blood lead. For example, the blood-lead level of the 0-3 year age group was 11.9 $\mu\text{g/dL}$ while that of the 4-6 year age group was 15.5 $\mu\text{g/dL}$. In other words, no peak value at 2-3 years was observed.

It is of course quite possible that children in hospital eat less, and so take in less lead, than comparable healthy children. Presumably, this would tend simply to lower the blood-lead levels in all age groups. The peak value at 2-3 years would show up even in hospitalized children if it were a basic characteristic of this age. It does not. Moreover, in the

two studies (11, 12) where measurements were made on both hospitalized and normal individuals, there is a smooth transition between the blood-lead levels of the two groups (Fig. 4 and 5).

It is difficult to make theoretical estimates of the blood-lead levels of children. The ratio of the intakes of air and food per unit blood volume of young children compared with women is about 1.5 (19). But it does not follow that the ratio of blood-lead concentrations (child/mother) to be expected is also 1.5, because there are other parameters—notably the fraction of ingested lead which is transferred to blood, and the clearance rate of lead from blood—which probably vary with age. There are no measured values of the half-life of lead in blood for children and very few measurements of gut absorption. But even if calculations of blood-lead levels are made with assumed values of these and other parameters (food intake, air intake, concentrations of lead in food and in air, blood volume, etc.) it requires curious assumptions to produce a curve of blood-lead versus age which shows a peak in early childhood.

A plausible working hypothesis is, then, that if lead in diet and in air are the only significant contributors to total lead intake, the concentration of lead in blood increases smoothly from the first year of life until at least middle age; during adolescence, the increase is much more marked for males than for females. The occurrence of child/woman blood-lead ratios greater than unity (more accurately, greater than some number less than unity) indicates an important pathway of lead uptake affecting children and not adults.

Child/Adult Blood-Lead Ratios in Areas of High and Low Contamination

Those studies in which investigators have compared two or more groups of people exposed to different levels of lead contamination are of interest. If the uptake of lead in dust is important for children, but less so for adults, then the child/adult blood-lead ratio might be expected to be higher in the more contaminated area.

Elwood et al (20) measured the blood-lead levels of mothers and children (0-5 years) living in the vicinity of a large battery factory. They studied two groups: leadworkers' families, and randomly selected controls. A summary of their capillary-blood measurements is given in Table 4. The child/mother blood-lead ratio is greater for the group exposed to the higher contamination.

Johnson et al (21) studied two populations in

Table 3. Blood-lead levels of three groups of children.^a

Group	Blood-lead level (mean \pm SD), $\mu\text{g/dL}$	No. of children >40 $\mu\text{g/dL}$
A	29.6 \pm 18.6	3
B	16.4 \pm 9.8	0
C	32.4 \pm 30.3	6

^a Data of Gibson et al. (17).

Table 4. Blood-lead levels of mothers and children.^a

Group	Mean blood-lead levels, $\mu\text{g/dL}$		Ratio child/mother
	Children	Mothers	
Leadworkers' families	33.1	23.6	1.40
Control families	27.0	21.9	1.23

^a Data of W. Elwood et al. (20).

California, one living near a Los Angeles freeway, the other at Lancaster in a desert area. The populations were divided into three age groups: group I (0-16 years), group II (17-34 years) and group III (35 years and over), and males and females were considered separately. The authors expressed the measured blood-lead values in terms of both arithmetic and geometric means; the latter are used in this discussion. Some of their results are given in Table 5. The child/woman ratio is greater in the more exposed group; the effect is rather more pronounced if the arithmetic means are used. It is possible to make estimates of α from these data: α is defined as the increase, $\Delta\text{Pb(B)}$, in blood-lead level which arises from an increase, $\Delta\text{Pb(A)}$, in the concentration of airborne lead. If it is assumed that the difference between the Los Angeles and Lancaster blood-lead levels is due solely to the higher intakes of inhaled lead by the Los Angeles groups, then the value of α for male children is 1.8, for female children it is 0.9 and for adults (group II males and females combined), it is 0.6. It seems unlikely that such differences in α really exist, and the assumption that inhalation is the only important route for children could therefore be incorrect.

In a study carried out around a lead smelter in Belgium, Roels et al. (22) measured the blood-lead levels of children (9-14 years) and some teachers at two schools within 1 km of the smelter, and also at two control schools. The relevant results are summarized in Table 6. The child/teacher ratio is greater for the schools nearer the smelter. A possible explanation might be that the teachers at the 'near smelter' schools tended to live further away from the smelter than the children and so were exposed to lower average concentrations of airborne lead. The value of α for the teachers (estimated from Table 6) is about 2—the commonly accepted value—which suggests that they were exposed to the concentrations of airborne lead quoted in Table 6 for most of the time. The value of α for the children (again, estimated from Table 6) is about 7. This is an unusually high value and suggests that, either the children were affected by an additional route of lead to the body, or their homes were much nearer the smelter than were the

Table 5. Levels of air and blood lead in California.^a

Area	Air lead, $\mu\text{g}/\text{m}^3$	Mean blood-lead level, $\mu\text{g}/\text{dL}$					Ratio child (M & F)/ woman
		Group I			Group II		
		M	F	M&F	M	F	
Los Angeles	6.3	20.8	14.9	18.0	15.1	11.8	1.53
Lancaster	0.64	10.4	9.6	10.7	10.9	8.0	1.34

^a Data of Johnson et al. (21).Table 6. Air and blood-lead levels near a Belgian smelter.^a

Type of school	Air lead, $\mu\text{g/m}^3$	Mean blood-lead level, $\mu\text{g/dL}$		Ratio, child/ teacher
		Children	Teachers	
Near smelter	2.68	27.8	18.0	1.54
Control	0.46	11.7	12.8	0.91

^a Data of Roels et al. (22).Table 7. Blood-lead levels of mothers and children^a

Soil lead content	Mean blood-lead level, $\mu\text{g/dL}$		Ratio, child/mother
	Children	Mothers	
High	29.0	14.2	2.0
Medium	13.8	18.7	1.3
Low	20.7	14.1	1.5

^a Data of Barltrop et al. (24).

schools. This latter explanation seems unlikely from the authors' description of the study area (23).

Barltrop et al. (24) measured the blood-lead levels of mothers and children exposed to lead contaminated soils and dusts in a rural area of Derbyshire with minimal atmospheric pollution. The data were classified into three groups depending on the soil-lead content found at the child's home and are summarized in Table 7. The child/mother ratios do not show a trend with soil-lead content.

Yankel et al. (25) carried out an extensive environmental survey around a large lead smelter in Idaho. They placed their study population into subgroups according to age and distance from the smelter; the region was divided into five areas (area I nearest to and area V furthest from the smelter), and there were two control areas. Some of the results are given in Table 8; their figures for 2 year-old and for 3-year old children have been pooled. The child/adult blood-lead ratio tends to decrease as the distance from the smelter increases, i.e., the ratio is enhanced for the more exposed groups.

In a study of environmental contamination around a lead smelter in London, Lansdown et al. (26) measured blood-lead levels in mothers and chil-

Table 8. Blood-lead levels of adults and children.^a

Region	Mean blood-lead level, $\mu\text{g/dL}$		Ratio, child/adult
	Children (2-3 yr)	Adults	
I (near smelter)	73.5	37	2.0
II	53	33	1.6
III	36	30	1.2
IV	34.5	34	1.0
V (far from smelter)	32	32	1.0
VI Control	23.5	—	—
VII Control	29	32	0.9

^a Data of Yankel et al. (25).

dren and grouped the results according to the distance of the homes from the factory. The figures in Table 9 are derived from their results. The child/mother ratio is higher for the group living nearer the factory.

Millar (27), in a study of contamination around another lead works in London, measured the blood-lead levels of both leadworkers' children and community children. He reported no measurements on adults, but since he grouped the children by age it is possible to compare the blood-lead levels of young (0-4 years) and older (11-16 years) children. His results for workers' children and for community children are summarized in Tables 10 and 11, respectively. In each case, the young child/old child blood-lead ratio is higher for the more exposed group.

Differences between the blood-lead levels of young and older children (but not adults) are also reported in a study carried out by Schmitt and his co-workers (28) around a smelter in Canada. They grouped their results by age and distance of home from the smelter as shown in Table 12. There is a consistent pattern. The young child/old child blood-lead ratio increases both with increasing contamination and with the gap between the two age groups. In another Canadian study, Roberts et al. (29) also measured the blood-lead levels of people living in the neighborhood of smelters. The authors did not report the data for children and adults separately, but they noted that "although there was no significant trend for adults, the blood-lead concentrations of the children increased with proximity to both smelters." It follows that the child/adult blood-lead ratio was higher for the more exposed groups.

Day et al. (30) looked at blood-lead levels in a group of people living close to a motorway immediately before and at two intervals after its opening. Some of their results are given in Table 13. Neither the absolute blood-lead levels, nor the child/woman ratios, show any trend with time. The traffic flows on this motorway (about 20,000 vehicles per 24 hr)

Table 9. Blood-lead levels of mothers and children^a

Distance from factory, m	Mean blood-lead level, $\mu\text{g/dL}$		Ratio, child/mother
	Children (5 yr)	Mothers	
<400	38	30	1.27
400-500	31	26	1.19

^a Data of Lansdown et al. (26).Table 10. Blood-lead levels of community and leadworkers' children.^a

Group	Mean blood-lead level, $\mu\text{g/dL}$		Ratio young/old
	Young children	Old children	
Leadworkers' children	27.4	17.5	1.57
Community children	21.1	15.6	1.35

^a Data of Millar (27).Table 11. Blood-lead levels of community children according to distance from factory.^a

Distance of home from factory, m	Mean blood-lead level, $\mu\text{g/dL}$		Ratio young/old
	Young children	Old children	
<280 m	22.4	15.6	1.44
280-400	19.7	15.6	1.26

^a Data of Millar (27).

were not particularly high, and their growth was to some extent balanced by falls in traffic flows on already existing roads.

Worth and her colleagues (31) measured the blood-lead levels of several hundred people in Massachusetts in an investigation designed to find the contributions made by tap water and other sources to lead intake. A minority of the study population were "referrals from the neighborhood health centres;" the authors do not indicate why referrals were made but presumably increased exposure to environmental lead was suspected. The relevant results are given in Table 14. The child/adult blood-lead ratio is greater in the more exposed group.

In almost all the studies mentioned in this section, the result is the same: the child/adult (or young child/old child) blood-lead ratio increases as the level of lead contamination increases. In all the studies, either all-venous or all-capillary blood sampling was used. No attempt has been made to calculate the errors associated with the individual ratios tabulated above, indeed it is not always possible to do so from the published data. However, it is unlikely that

Table 12. Soil and blood-lead levels near a Canadian smelter.^a

Distance from smelter, km	Soil lead, ppm	Mean blood-lead level of children, $\mu\text{g/dL}$			Ratio, youngest/oldest
		1-3 yr	ca. 6 yr	ca. 15 yr	
<1.6	1662	28.8	25.1	10.4	2.8
1.6-3.2	1341	26.6	24.6	11.8	2.3
>3.2	354	18.7	17.8	11.5	1.6

^a Data of Schmitt et al. (28).Table 13. Blood-lead levels of women and children near a motorway.^a

Time of measurements	Mean blood-lead level, $\mu\text{g/dL}$		Ratio child/woman
	Children (0-10 yr.)	Women	
Before opening	16.3	14.0	1.16
6 months after opening	15.4	13.9	1.11
1 year after opening	16.5	14.7	1.12

^a Data of Day et al. (30).

chance is responsible for the similar pattern of so many diverse studies.

Although it is probable that the fraction of ingested lead which is transferred to blood is higher for children than for adults (32, 33), there does not appear to be any evidence that this fraction—or any of the other relevant toxicokinetic parameters for children and adults—will vary with intake in such a way as to produce the enhancement of the child/adult blood-lead ratio observed in contaminated areas. Indeed, there is some evidence to the contrary from two different blood-lead studies of mothers and their children: that carried out by Elwood et al. (34) in Wales and that carried out on mothers and babies in Glasgow (10). In both studies, the mothers and their children had unusually high intakes of ingested lead because of high concentrations of lead in water. However, the child/mother blood-lead ratio was close to unity in each study (the numerical value of the ratio was the same in each study—1.05).

The results from most of the studies described in this section do therefore indicate a pathway to the body which is more important for children than for adults, but they provide no information as to its nature.

Possible Routes from the Environment to the Body

As was noted in the Introduction, the ingestion of lead in dust via the hands has frequently been suggested as an important pathway for young children. It might be inferred to be so from the most casual

Table 14. Blood-lead levels of adults and children in Massachusetts.^a

Type of case	Mean blood-lead level, $\mu\text{g/dL}$		Ratio, child/adult
	Children (2-6 yr)	Adults (20-50 yr)	
Referred	32.85	21.91	1.50
Nonreferred	23.99	18.99	1.26

^a Data of Worth et al. (31).

observation of young children's behavior and was discussed as long ago as 1904 by Gibson (35). However, other possible reasons for a high uptake of lead during early childhood have been put forward.

The value of α may be larger for young children than for adults. Children inhale more air relative to their body weight than do adults. If the fraction of inhaled lead transferred to blood, and the half-life of lead in blood are assumed to be the same for the child as for the adult then α for a 2-year-old child would be about 4 (36, 37). However, such estimates are of limited value, because there is no reason to believe that these two parameters are the same for child and adult; further, it seems unlikely that α would peak at 2-3 years of age. The results from some of the environmental studies make it difficult to accept that the enhanced blood-lead levels found in children are a consequence of a high value of α . For example, if the differences in blood lead reported by Schmitt et al. (28) were due solely to inhalation, a value of α equal to zero would have to be assumed for 15-year-old children, together with a very high value (probably about 7) for the youngest children. Again, high blood-lead values of leadworkers' children, which correlate well with lead in house-dust, have been reported by some authors. The levels of airborne lead are not usually given but are probably quite low. An explanation of the results based on inhalation would require the assumption of very large differences, $\Delta\text{Pb(A)}$, in the concentrations of airborne lead at different workers' houses, and/or very large values of α . For example, in the study reported by Baker et al. (38), the difference, $\Delta\text{Pb(B)}$, between the highest and lowest blood-lead groups was 43 $\mu\text{g/dL}$. Any combination of $\Delta\text{Pb(A)}$ and α which would give this value of $\Delta\text{Pb(B)}$ is scarcely believable. Finally, Walter et al. (39), in an examination of the data from their Silver Valley study, carried out an analysis to identify the important variables (in the prediction of children's blood-lead levels) and how these changed with age. They concluded that the effect of airborne lead was virtually constant for all age groups (1 year to 9 years of age), while the effect of household dustiness declined with age.

Some airborne lead is deposited onto food and

kitchen utensils. This entry route may well be responsible for a significant fraction of some people's total intake (40) but it is probably equally important for both children and adults and is unlikely to account for an enhanced child/adult ratio. It could however be partly responsible for an enhanced urban/rural ratio.

The resuspension of deposited lead and its subsequent inhalation has been mentioned as a factor which may be more important for young children than for adults (23, 28). It is difficult to judge the relative importance of this pathway, but there are some pointers. Thus Lepow et al. (41), in an attempt to resolve this problem, measured the concentration of airborne lead at two heights (2 ft and 5 ft) at each of four sites where their study children played. The differences between the concentrations at the two heights were not significant at any of the sites. Again, during the study mentioned earlier, Roels and his colleagues (22) measured not only airborne lead and blood-lead levels, but also the amount of lead, Pb(H), on the hands of the children. The authors found that the multiple regression coefficient of $Pb(B)/Pb(A)\log Pb(H)$ calculated for the four groups of children combined was highly significant. Standardization for Pb(A) resulted in a still significant partial correlation between Pb(B) and $\log Pb(H)$, whereas the partial correlation between Pb(B) and Pb(A) became insignificant when standardized for $\log Pb(H)$. The authors concluded that the ingestion of lead via dirty hands was a much more important factor contributing to Pb(B) than was Pb(A). A further finding of interest was that the boys had significantly higher Pb(H) levels and—like the boys in the study of Johnson et al. (21)—significantly higher Pb(B) levels, than did the girls. The average boy/girl ratios for these two parameters were about 2 and 1.2, respectively.

Several workers (41-44) have shown that measured values of the lead present on children's hands, combined with quite modest assumptions about the frequency of hand mouthing, suggest that many children are likely to ingest substantial quantities of lead (comparable with the amount taken in via diet) by the hand-to-mouth route. A study by Charney et al. (45) is of interest. About 50 children with high blood-lead levels (40-70 $\mu\text{g/dL}$) were matched with 50 children with low levels (less than 29 $\mu\text{g/dL}$), and housedust lead, lead on hands, and several other variables noted for each child. Children in the high blood-lead group showed significant differences from the low lead children in a number of ways: for example, they had higher hand-lead levels, their homes had higher dust-lead levels, they played more often in outside soil and their soil had a higher lead content, they mouthed

objects more often and they sucked their fingers more often.

There are, then, some grounds for believing that the ingestion of lead in dust, sometimes soil, via the hands is the pathway mainly responsible for the enhanced child/adult blood-lead ratio so frequently observed.

Quantitative Relationships

A review of those published studies from which a quantitative relationship between children's blood-lead levels and the concentration of lead in dust or soil can be inferred has been carried out (2). It was concluded that the factor δ , defined as the increment, $\Delta Pb(B)$ in blood-lead level associated with an increment, $\Delta Pb(D)$ in the concentration of lead in dust or soil, varied from about 1 to 10 $\mu\text{g/dL}/1000$ ppm, with a median value of about 5 $\mu\text{g/dL}/1000$ ppm. The increase in blood-lead level indicated by this relationship is additional to any increase due to airborne lead.

Since that review, the Department of the Environment has reported the UK results for the first campaign of the EEC screening program for lead (10). The report details blood-lead measurements made on children living in the vicinity of smelters and on children living near busy roads. A report on measurements of environmental lead near some of these works and roads has also been published (46). It was therefore anticipated that estimates of δ could be derived from all these data, but this has not proved possible. There is not sufficient cross-referencing between the blood lead and the air and dust lead measurements, and the dust sampling was confined to gutters of major roads, together with a very few house interiors. A possible exception is provided by the data from the Greenwich Survey. More children (400 in all) were examined here than any of the other locations, and additional environmental measurements are available from another survey (47). However, the two surveys were carried out at different times, and the area around the lead works was subdivided in different ways. It is therefore difficult to combine the dust and blood-lead data properly, but a low value of δ —probably about unity—is indicated.

The investigation carried out by Barltrop et al. (24) in Derbyshire is often cited as a study which shows that, although lead in soil and dust does have some effect upon blood lead, the effect is a small one. The relevant results are given in Table 15. The values of δ obtained from these data are about 0.6 and 4 $\mu\text{g/dL}/1000$ ppm for the lead in soil and lead in dust, respectively. Since 2-year-old children spend more of their waking hours indoors than in the

Table 15. Mean levels of lead in soil, dust and blood in three regions in Derbyshire.^a

Lead in soil, ppm	Lead in housedust, ppm	Lead in children's blood, $\mu\text{g/dL}$
420	531	20.7
3390	1564	23.8
13969	2582	29.0

^a Data of Barltrop et al. (24).

garden, the value of δ for housedust is the one of greater significance. This value of $4 \mu\text{g/dL}/1000 \text{ ppm}$ is close to the median value ($5 \mu\text{g/dL}/1000 \text{ ppm}$) obtained from the review.

Some work by Rice and her colleagues (48) was not used in the review because they measured zinc protoporphyrin (ZPP) and not blood-lead levels in their study children. However, in one of the studies which was used—that by Watson et al. (49)—the authors measured both blood-lead and ZPP levels. Their blood-lead measurements gave a value for δ of about $7 \mu\text{g/dL}/1000 \text{ ppm}$ —fairly close to the median value; their ZPP measurements give a value for $\delta(\text{ZPP})$ of about $9 \mu\text{g ZPP/dL}/1000 \text{ ppm}$. The value of $\delta(\text{ZPP})$ estimated from the results of Rice et al. is about the same— $11 \mu\text{g ZPP/dL}/1000 \text{ ppm}$. Erythrocyte protoporphyrin varies exponentially with blood-lead level and it may therefore be more appropriate to work in terms of the logarithm of the ZPP level. The values of $\delta (\ln \text{ ZPP})$ obtained from the Watson and the Rice studies are 0.26 and 0.23, respectively. Hence, the work of Rice et al. suggests a quantitative relationship (between body lead and lead in dust) similar to the one derived in the review.

Another study not included in the review is that carried out by Schmitt et al. (28); their measurements are summarized in Table 13. The authors quote only one measurement of airborne lead: the annual concentration in the region nearest the smelter was $2 \mu\text{g/m}^3$. However, it seems unlikely that airborne lead had much effect on the mean blood-lead levels given in Table 12; if it had had, one would expect a decrease in the 15-year-olds' blood-lead levels with increasing distance from the smelter. On the assumption that airborne lead was not influential in determining the differences in blood-lead levels observed, then the value of δ estimated from Table 12 for 1 to 3-year-old children is about $8 \mu\text{g/dL}/1000 \text{ ppm}$.

Finally, some recent work by Milar and Mushak (50) is of relevance here. In one part of their study the authors report a decline in the blood lead of a 2-year-old child over a 9-month period during which lead-decontamination procedures (principally, the thorough cleaning of carpets) were carried out on

three occasions at the child's home. During this period, the level of lead in house dust fell from about 8000 to 2000 ppm, and the child's blood-lead level fell from about 50 to $20 \mu\text{g/dL}$. This is only one case, but the concomitant decline in the values of the two parameters is suggestive; the value of δ from this part of the study is $5 \mu\text{g/dL}/1000 \text{ ppm}$. In another part of the study, measurements were made of the concentration of lead in house dust and of the blood-lead levels of 17 children between the ages of 1.5 and 4 years who were exposed via a parent to lead from industrial sources; the children had an average blood-lead level of $44 \mu\text{g/dL}$, and the concentration of lead in the house dust of their homes was about 3000 ppm. A group of control children of the same age, but not exposed to lead from industrial sources or from lead-based paint, had an average blood-lead level of $18 \mu\text{g/dL}$ and the average concentration of lead in house dust was about 250 ppm. A comparison of these two groups suggests a value for δ of about $9 \mu\text{g/dL}/1000 \text{ ppm}$. There seems no reason to change the main conclusion (given at the beginning of this section) reached in the review.

Discussion

There is a *prima facie* case for the hypothesis that the ingestion of urban dust via dirty hands gives rise to a significant intake of lead during early childhood. The peak level of blood lead frequently observed at 2-3 years of age, the enhancement of the child/adult blood-lead ratio which occurs in contaminated areas and the relationship between blood lead and lead in dust seen in many studies, can all be accounted for by the hypothesis.

For most urban children it is likely that a greater intake of lead comes about from the ingestion of dust than from the inhalation of airborne material. It follows that there is a need for a lead-in-dust standard to be used in parallel with a lead-in-air one. It is sometimes said that a lead-in-dust standard is impractical because the monitoring required presents such difficulties: principally those arising from large spatial and temporal variations in the concentration of lead in dust. However, while it is true that the taking of a few spot samples can give a very misleading picture, the setting up of an effective monitoring program is quite practicable, particularly if large area sampling is used (51). It should be noted here that analogous difficulties of monitoring (52) have not prevented the adoption of standards for airborne lead.

Perhaps there is also the view that if δ were really as high as $5 \mu\text{g/dL}/1000 \text{ ppm}$ then one might expect a greater difference in the blood-lead levels of

urban and rural children than is actually observed. But there are few studies comparing matched populations of children from urban areas with those from rural (or suburban) areas on which to base such a view. And what studies there are do not present a uniform picture. The measurements made by Taskinen and her colleagues (16) of blood-lead levels of Finnish preschool children show a very low mean value (about 6 $\mu\text{g/dL}$), no correlation with age, and no significant differences in the blood-lead levels of children from three different types of area (urban, rural and smelter); the authors mention an annual mean concentration of airborne lead of 0.94 $\mu\text{g/m}^3$ at one of the busiest city-center crossings in Helsinki, but otherwise give no data on air and dust lead in their survey areas. A recent study by Elwood et al. (53) among preschool children in Wales showed no difference in the mean blood-lead level of a group living adjacent to busy major roads compared with that of a group living in a rural area. By contrast, some work from the United States (54-56) suggests that the blood-lead levels of urban children may be about 10 $\mu\text{g/dL}$ higher than those of rural children (such a large difference could not be accounted for by the inhalation route alone) although it must be noted that there were difficulties in matching for race. Again, in a study of urban and suburban school children (aged 10-15 years) in Tokyo, Okubo et al. (57) found significantly higher blood-lead levels in the urban groups. The blood-lead levels of the urban children decreased with age while those of the suburban were roughly constant; the urban/suburban difference therefore decreased with age (from about 5.5 $\mu\text{g/dL}$ for the 10-year-olds to about 1.5 $\mu\text{g/dL}$ for the 15-year-olds). The authors report similar concentrations of airborne lead in the urban and suburban areas (about 0.55 $\mu\text{g/m}^3$ and 0.4 $\mu\text{g/m}^3$, respectively) and speculate that exposure to lead in street dust is probably responsible for the differences in blood-lead levels.

There is a general paucity of data on lead-in-dust concentrations in rural communities. Levels in outer suburbs or in medium-sized towns are sometimes not much less than in inner-city areas (42, 58). It would be instructive to have blood-lead data on children from urban and rural areas where comprehensive measurements of environmental lead levels have shown a marked urban/rural difference.

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